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(FILE 'HOME' ENTERED AT 11:17:14 ON 18 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 11:17:37 ON 18 APR 2007

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L1           364 S (MYOFILAMENT PROTEIN)
L2             2 S L1 AND (MUSCLE DAMAGE)
L3             2 DUPLICATE REMOVE L2 (0 DUPLICATES REMOVED)
L4          6191 S (MUSCLE DAMAGE)
L5             2 S L1 AND L4
L6            29 S L4 AND ADDUCT?
L7             4 S L6 AND TROPONIN?
L8             3 S L7 NOT L5
L9             1 DUPLICATE REMOVE L8 (2 DUPLICATES REMOVED)
L10           13 DUPLICATE REMOVE L6 (16 DUPLICATES REMOVED)
L11           12 S L10 NOT L5
L12           11 S L11 NOT L9
L13            3 S L12 AND PD<2000
L14            8 S L12 NOT L13
L15         4543 S (PROTEIN ADDUCT)
L16            6 S L15 AND L4
L17            2 DUPLICATE REMOVE L16 (4 DUPLICATES REMOVED)
L18          202 S L4 AND TROPONIN?
L19           93 S L18 AND PD<2000
L20           47 DUPLICATE REMOVE L19 (46 DUPLICATES REMOVED)
L21            0 S L20 AND ADDUCT?
L22            2 S L20 AND TNI?
L23            0 S L20 AND TNC
L24            0 S L20 AND TNC?
L25            0 S (TNI? ADDUCT)
L26            0 S (TROPONIN ADDUCT?)
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L23            0 S L20 AND TNC
L24            0 S L20 AND TNC?
L25            0 S (TNI? ADDUCT)
L26            0 S (TROPONIN ADDUCT?)

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ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2

AN 2000:809166 CAPLUS

DN 134:111508

ED Entered STN: 19 Nov 2000

TI Experimental heart muscle damage in alcohol feeding is associated with increased amounts of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts

AU Worrall, Simon; Richardson, Peter J.; Preedy, Victor R.

CS Alcohol Research Unit, Department of Biochemistry, The University of Queensland, Brisbane, QLD 4072, Australia

SO Addiction Biology (2000), 5(4), 421-427
CODEN: ADBIFN; ISSN: 1355-6215

PB Carfax Publishing

DT Journal

LA English

CC 4-7 (Toxicology)

AB Chronic and excessive alc. consumption induces defined myocardial lesions characterized by impaired structural, mech. and biochem. features. The pathogenic mechanisms are unknown, although it is possible that protein adduct formation by reactive metabolites of ethanol may be a contributory process. Hitherto, this has only been tested with respect to antibodies against reduced-acetaldehyde protein adducts in clin. studies, despite the fact that during alc. toxicity the formation of reduced-acetaldehyde, unreduced-acetaldehyde, malondialdehyde, malondialdehyde-acetaldehyde and hydroxyethyl protein adducts have been reported in non-cardiac tissues. It was the author' hypothesis that the heart is particularly sensitive to the formation of protein adducts in alc. toxicity. To test this hypothesis, the authors analyzed hearts from rats fed nutritionally complete liquid diets containing ethanol as 35% of total calories for 6 wk, using the Lieber-DeCarli pair-feeding protocol. Control rats were treated identically and fed the same diet in which ethanol was replaced by isocaloric glucose. At the end of the feeding period, the hearts were dissected and ventricular muscle analyzed. After 6 wk' ethanol feeding, ELISA anal. showed increased amts. of reduced-acetaldehyde protein adducts ($p < 0.01$) unreduced-acetaldehyde ($p < 0.01$) and malondialdehyde-acetaldehyde ($p = 0.01$) protein adducts. However, malondialdehyde and α-hydroxyethyl- protein adducts were not significantly increased in hearts of ethanol-fed rats compared to pair-fed control ($p > 0.1$ in both instances). This is the first report of acetaldehyde adduct formation in alc. cardiomyopathy. This suggests that either immune process may develop or functional impairment of affected proteins may occur.

ST heart muscle damage alc acetaldehyde malonaldehyde protein adduct; ethanol cardiotoxicity oxidative stress heart acetaldehyde malonaldehyde protein adduct; lipid peroxidn heart acetaldehyde malonaldehyde protein adduct

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(adducts; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT Heart, disease
(cardiomyopathy; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT Toxicity
(cardiotoxicity; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with

increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT Immunoassay
(enzyme-linked immunosorbent assay; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT Heart
Immunity
Oxidative stress, biological
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT Peroxidation
(lipid; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT Heart
(toxicity; ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT 64-17-5, Ethanol, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT 75-07-0, Acetaldehyde, biological studies
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

IT 75-07-0D, Acetaldehyde, adducts with proteins, biological studies
542-78-9D, Malondialdehyde, adducts with proteins
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(ethanol cardiotoxicity in relation to exptl. heart muscle damage in alc. feeding is associated with increased amts. of reduced- and unreduced-acetaldehyde and malondialdehyde-acetaldehyde protein adducts)

RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD

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ANSWER 1 OF 2 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1996:62716 BIOSIS
DN PREV199698634851
TI Myosin heavy-chain fragments and cardiac troponins in the serum in rhabdomyolysis: Diagnostic specificity of new biochemical markers.
AU Lofberg, Mervi [Reprint author]; Tahtela, Riitta; Harkonen, Matti; Somer, Hannu
CS Dep. Neurol., Helsinki Univ. Central Hosp., 00290 Helsinki, Finland
SO Archives of Neurology, (1995) Vol. 52, No. 12, pp. 1210-1214.
CODEN: ARNEAS. ISSN: 0003-9942.
DT Article
LA English
ED Entered STN: 9 Feb 1996
Last Updated on STN: 10 Feb 1996
AB Background: Myosin is the major structural protein in muscle. Antibodies to beta-type heavy meromyosin react with cardiac and slow-twitch skeletal muscle. Cardiac TnT and TnI were developed as tissue-specific indicators. Objectives: To study myosin heavy-chain fragments as a delayed marker of previous rhabdomyolysis. To examine the cardiac specificity of cardiac troponin T (TnT) and cardiac troponin I (TnI) in patients with severe skeletal muscle damage. Design and Methods: Serum myosin heavy-chain fragments, TnT, and TnI were studied up to 12 days after diagnosis in relationship to the serum creatine kinase level in 20 patients with rhabdomyolysis. The mean peak serum creatine kinase activity was 91 300 U/L. Myosin heavy-chain fragments were measured by an immunoradiometric assay, TnT by a one-step immunoenzymometric assay, and TnI by an immunoenzymometric assay. Results: Values for serum myosin heavy-chain fragments were greater than the upper limit of normal in all patients. The peak value (70 times the upper normal limit, on average) was usually achieved 4 to 7 days after the diagnosis of rhabdomyolysis, and it was increased up to 12 days. The peak level of TnT was increased in 95% of the patients, and it correlated strongly with the peak activity of serum creatine kinase. The highest Tnl value was above the detection limit of myocardial infarction in 30% of the patients. Half of these patients were the only patients with ischemic changes observed on an electrocardiogram performed on admission to the hospital. Conclusions: The measurement of myosin heavychain fragments was useful in the diagnosis of previous rhabdomyolysis up to 12 days. The role of TnT was negligible as an indicator of cardiac muscle damage in patients with severe rhabdomyolysis. Cardiac TnI is a more tissue-specific marker for myocardial damage even with concurrent rhabdomyolysis.
CC Mathematical biology and statistical methods 04500
Clinical biochemistry - General methods and applications 10006
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Enzymes - Physiological studies 10808
Pathology - Diagnostic 12504
Metabolism - Minerals 13010
Metabolism - Proteins, peptides and amino acids 13012
Cardiovascular system - Physiology and biochemistry 14504
Cardiovascular system - Heart pathology 14506
Cardiovascular system - Blood vessel pathology 14508
Blood - Blood and lymph studies 15002
Blood - Blood, lymphatic and reticuloendothelial pathologies 15006
Muscle - Physiology and biochemistry 17504
Muscle - Pathology 17506
Nervous system - Physiology and biochemistry 20504
Nervous system - Pathology 20506
Public health - Public health administration and statistics 37010
Public health - Health services and medical care 37012
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cardiovascular

Medicine (Human Medicine, Medical Sciences); Cardiovascular System (Transport and Circulation); Clinical Chemistry (Allied Medical Sciences); Enzymology (Biochemistry and Molecular Biophysics); Hematology (Human Medicine, Medical Sciences); Mathematical Biology (Computational Biology); Metabolism; Muscular System (Movement and Support); Nervous System (Neural Coordination); Neurology (Human Medicine, Medical Sciences); Pathology; Public Health (Allied Medical Sciences)

IT Chemicals & Biochemicals
CREATINE KINASE

IT Miscellaneous Descriptors
BETA-TYPE HEAVY MEROMYOSIN; CARDIAC MUSCLE DAMAGE; CARDIAC TROPONIN I; CARDIAC TROPONIN T; CREATINE KINASE; ISCHEMIA; MAJOR STRUCTURAL PROTEIN; MYOCARDIAL INFARCTION; NEUROLOGY; SEVERE SKELETAL MUSCLE DAMAGE; SLOW-TWITCH SKELETAL MUSCLE; STATISTICAL ANALYSIS

ORGN Classifier
Hominidae 86215

Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia

Organism Name
human

Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

RN 9001-15-4 (CREATINE KINASE)

AN 1998152608 EMBASE
TI Cardiac troponin-I is a marker of perioperative myocardial damage during cardiac surgery.
AU Contini G.A.; Albertini D.; Antonelli A.M.; Campodonico R.; Corsanini E.; Grattagliano C.; Reverberi C.; Medici D.; Contini S.A.; Fragnito C.; Barboso G.
CS Dr. G.A. Contini, Divisione Cartedra Cardiochirurgia, Azienda Ospedaliera di Parma, Via Gramsci 14, 43100 Parma, Italy
SO Journal of Cardiovascular Diagnosis and Procedures, (1998) Vol. 15, No. 1, pp. 41-45. .
Refs: 15
ISSN: 1073-7774 CODEN: JCDPE
CY United States
DT Journal; Article
FS 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 2 Jun 1998
Last Updated on STN: 2 Jun 1998
AB Diagnosis of perioperative myocardial infarction is a challenging task. Clinical symptoms, electrocardiographic changes, and biochemical markers have inherent limitations as diagnostic tools; isoenzymes of creatine kinase, for instance, are of limited specificity because of skeletal muscle damage. Cardiac troponin 1 (cTnI) is considered by many investigators to be more cardiospecific than CK-MB. The aim of this study was to assess, with serial measurements of levels of cTnI and mass CK-MB, the incidence and importance of cardiac injury and its relation to the length of extracorporeal circulation and aortic cross-clamp time in patients undergoing cardiac surgery. Serial measurements of cTnI using the Stratus® TnI assay which employs monoclonal antibodies specific for the TnI cardiac isotype and mass CK-MB have been performed in seventy consecutive patients operated on for coronary bypass surgery or valve replacement. All the patients have been operated on by different surgeons, with standard extracorporeal and surgical procedures; myocardial protection was achieved by hematic cardioplegy, either cold intermittent or warm intermittent or continuous. After operation they were admitted in a dedicated intensive care unit with monitoring facilities. Baseline measurements of cTnI were below 0.6 ng/ml (0.129 ± 0.422), and rose slightly in normally convalescing patients, with peak values between 6 hours and the first postoperative day. Serum levels of cTnI rose in patients with myocardial injury and in those with longer extracorporeal circulation and aortic cross-clamp times.
CT Medical Descriptors:
*heart muscle injury: CO, complication
*heart muscle injury: DI, diagnosis
*extracorporeal circulation
*aorta clamping
*heart surgery
coronary artery bypass surgery
heart valve replacement
time
heart protection
cardioplegia
heart disease: SU, surgery
heart infarction: CO, complication
heart infarction: DI, diagnosis
human
male
female
major clinical study